Notices

Organisers of meetings who wish to insert notices should send details to the editor (address on the inside front cover) at least eight months before the date of the meeting or six months before the closing date for application.

Sixth Forum of International Andrology

The 6th forum of international andrology will be held on Tuesday 3 and Wednesday 4 May 1988 at the Intercontinental Hotel, 3 rue de Castiglione, 75001 Paris, France.

Topics will include:

Round tables on: prostatic adenoma: physiopathology, oligo-astheno-teratozoospermia, and priapism;

Workshops on: Total prostatectomy: techniques and indications, and premature and retrograde ejaculation: treatment;

Symposiums on: the principles of antibiotherapy in andrology, and the male body image;

What's new in andrology? (posters); and

Film and video session.

The official languages will be French and English and there will be simultaneous translations. The final programme will be available on request in April 1988. For further information please contact: Professor G ARVIS, Department of Andrology-Urology, Hopital Saint-Antoine, 184 rue du Faubourg Saint-Antoine, 75012 Paris, France Tel: (1) 43.4373.40; Télex: ARVIS 250 303 Public Paris.

Anglo-Scandinavian conference on sexually transmitted diseases

The Anglo-Scandinavian conference on sexually transmitted diseases will be held on 11 to 13 May 1988 at the Royal Society of Medicine, London.

For information please contact the conference secretariat (Miss Barbara Komoniewska) at the Royal Society of Medicine, 1 Wimpole Street, London W1 (Tel: 01 408 2119 ext: 301).

First conference of the European Society for Chlamydia Research

The first conference of the European Society for Chlamydia Research will be held on 30 May to 1 June 1988 in Bologna, Italy. The main topics will include: epidemiology and preventive measures against chlamydial infections (with *C trachomatis* and *C psittaci*) in Europe, biology, clinical manifestations and treatment, immunology and interaction between host and parasite, diagnostic procedures, chlamydial genetics, and vaccine development.

Please contact Dr Roberto Cevenini, Institute of Microbiology, University of Bologna, S Orsola University Hospital, 9 via Massarenti, 40138 Bologna, Italy (tel: 051-341652/302435).

34th General assembly of the International Union against the Venereal Diseases and Treponematoses (IUVDT)

The 34th general assembly of the IUVDT will be held on 3–4 June 1988 at the Palais des Festivals de Cannes, Cannes, France.

For information please contact Dr MA Waugh, Secretary General of the IUVDT, The General Infirmary at Leeds, Great George Street, Leeds LS1 3EX. (Tel: 0532 432799).

Spring meeting of the Medical Society for the Study of Venereal Diseases (MSSVD)

The spring meeting of the MSSVD will be held on 30 June to 3 July 1988 at Cambridge, England.

For information contact the local organiser, Dr MR FitzGerald, Addenbrooke's Hospital, Cambridge, CB2 2QQ or the travel agent, Mr Peter Davidson, Horncastle Travel Limited, 10 Market Street, Newcastle-upon-Tyne, NE1 6JF.

Second international lesbian and gay health conference

The second international lesbian and gay conference and AIDS forum will be held on 20–26 July 1988 at the Boston Park Plaza Hotel and towers in Boston, Massachusetts, USA. The conference is sponsored by the British Gay Medical Association and the American National Lesbian and Gay Health Foundation, the American Association of Physicians for Human Rights, and the George Washington University Medical Centre.

The overall goal of the conference is to constitute an international and national agenda for the next decade and will include topics such as sexual health, mental health issues, and holistic health care.

Further details can be obtained from: NLGHF/AAPHR Programming Committee, P O Box 65472, Washington DC 20035, USA.

Australian and New Zealand conference on sexually transmitted diseases

An Australian and New Zealand conference on sexually transmitted diseases will be held on 25 to 27 August 1988 at the University of Melbourne, Melbourne, Victoria, Australia.

For further information please contact: The Manager, National Australia Bank Ltd Travel Groups/Incentives, 271 Collins Street, Melbourne, Victoria, Australia 3000.

Courses on the acquired immune deficiency syndrome (AIDS)

The Royal College of Physicians of London is organising courses to train general physicians who will be concerned in the care of patients with AIDS. Each course will last for one week (Mondays to Fridays); mornings will be spent at the College and afternoons at one of four hospitals with major AIDS centres in London (St George's, St Mary's, St Stephen's, and the Middlesex). Numbers on each course will be limited to 20, with groups of five attending each hospital. The fee will be £90, and buffet lunch at the college each day and coffee or tea are included.

Starting dates and closing dates for applications are as follows:

Week starting .1988	*Closing date for applications*
5 September	26 July
21 November	10 October

For further details and application form, please contact: The Assistant Registrar, Royal College of Physicians, 11 St Andrew's Place, Regent's Park, London NW1 4LE (tel: 01 935 1174).

Institut Alfred Fournier Prix de l'Association des Anciens Élèves et Compagnons, 1988.

Deux prix d'un montant de fr 15 000 chacun destinés à récompenser un travail original ou un ensemble de travaux, dans le domaine des maladies transmises par voie sexuelle (MST),

—l'un en sciences fondamentales

—l'autre concernant le ou les sujets suivants: Épidémiologie—Biologie—Clinique—Thérapeutique

Les candidats devront adresser le texte de leur travail définitif, dactylographié et rédigé en français, présenté sous forme d'une publication, en six exemplaires, avant le 15 Septembre 1988.

La remise solennelle des Prix 1988 se fera lors de l'Assemblée Générale de l'Association des Anciens Élèves et Compagnons d'Alfred Fournier, en Novembre 1988,

Pour toute demande de renseignements et envoi de candidature, s'adresser au:
Secrétariat de l'Association, Institut Alfred Fournier, 25 Boulevard Saint-Jacques, 75680 PARIS CEDEX 14, (Tel: (1) 45 65 27 77).

List of current publications

Selected abstracts and titles from recent reports published worldwide are arranged in the following sections:

Syphilis and other treponematoses

Ultrastructural changes of *Treponema pallidum* isolated from secondary syphilitic skin lesions
A POULSEN, T KOBAYASI, L SECHER, K WEISMANN (Copenhagen, Denmark). *Acta Derm Venereol (Stockh)* 1987;**67**:289–94.

Changes in the cell surface properties of *Treponema pallidum* that occur during in vitro incubation of freshly extracted organisms
LV STAMM, RL HODINKA, PB WYRICK, PJ BASSFORD (Chapel Hill, USA). *Infect Immun* 1987;**55**:2255–61.

Activation of the classical and alternative pathways of complement by *Treponema pallidum* subsp *pallidum* and *Treponema vincentii*
TJ FITZGERALD (Duluth USA). *Infect Immun* 1987;**55**:2066–73.

Enzyme-linked immunosorbent assay for detection of antibodies to the venereal disease research laboratory (VDRL) antigen in syphilis
NS PEDERSEN, O ØRUM, S MOURITSEN (Copenhagen, Denmark). *J Clin Microbiol* 1987;**25**:1711–6.

Detection of *Treponema pallidum* by a fluorescent monoclonal antibody test
B ROMANOWSKI, E FORSEY, E PRASAD, S LUKEHART, M TAM, EW HOOK (Edmonton, Canada). *Sex Transm Dis* 1987;**14**:156–9.

Gonorrhoea

Auxotype/serovar diversity and antimicrobial resistance of *Neisseria gonorrhoeae* in two mid-sized American cities
EW HOOK, FN JUDSON, HH HANDSFIELD, JM EHRET, KK HOLMES, JS KNAPP (Atlanta, USA). *Sex Transm Dis* 1987;**14**:141–6.

Study of penicillinase producing *Neisseria gonorrhoeae* (PPNG) isolated in France from 1979 to 1986 by a multicentric group
JY RIOU, MF PRERE, Y PEAN, *et al* (Paris, France). *Pathol Biol (Paris)* 1987;**35**:791–5.

Antigenic and structural differences among six proteins II expressed by a single strain of *Neisseria gonorrhoeae*
DS BARRITT, RS SCHWALBE, DG KLAPPER, JG CANNON (Chapel Hill, USA). *Infect Immun* 1987;**55**:2026–31.

Effect of spectinomycin use on the prevalence of spectinomycin-resistant and of penicillinase-producing *Neisseria gonorrhoeae*
JW BOSLEGO, EC TRAMONT, ET TAKAFUJI, *et al* (Washington, USA). *N Engl J Med* 1987;**317**:272–8.

Non-specific genital infection and related disorders (chlamydial infections)

Male fertility and positive chlamydial serology: a study of 61 fertile and 82 subfertile men
MR AUROUX, DM de MOUY, JF ACAR (Kremlin Bicetre, France). *J Androl* 1987;**8**:197–200.

The immunobiology of *Chlamydia*
D LEVITT, J BAROL (Nutley, USA). *Immunology Today* 1987;**8**:246–51.

Specific effect of estradiol on the genital mucosal antibody response in chlamydial ocular and genital infections
RG RANK, AL BARRON (Little Rock, USA). *Infect Immun* 1987;**55**:2317–9.

The effects of contraceptive hormones on the replication of *Chlamydia trachomatis* in human endometrial cells
D KLEINMAN, I SAROV, V INSLER (Beer Sheva, Israel). *Contraception* 1987;**35**:533–42.

The use and limitations of endocervical Gram stains and mucopurulent cervicitis as predictors for *Chlamydia trachomatis* in female adolescents
B MOSCICKI, M-A SHAFER, SG MILLSTEIN, CE IRWIN, J SCHACHTER (San Franscisco, USA). *Am J Obstet Gynecol* 1987;**157**:65–71.

Should tests for *Chlamydia trachomatis* cervical infection be done during routine gynecological visits? An analysis of the costs of alternative strategies
RS PHILLIPS, MD ARONSON, WC TAYLOR, C SAFRAN (Boston, USA). *Ann Intern Med* 1987;**107**:188–94.

Use of a direct fluorescent antibody test for detecting *Chlamydia trachomatis* cervical

infection in women seeking routine gynecologic care
RS PHILLIPS, PA HANFF, RS KAUFFMAN, MD ARONSON (Boston, USA). *J Infect Dis* 1987;**156**:575–81.

Detection of *Chlamydia trachomatis* cervical infection: a comparison of Papanicolaou and immunofluorescent staining with cell culture
TC QUINN, PK GUPTA, RT BURKMAN, EW KAPPUS, M BABACCI, MR SPENCE (Bethesda, USA). *Am J Obstet Gynecol* 1987;**157**:394–9.

Effect of prior sexually transmitted disease on the isolation of *Chlamydia trachomatis*
BP KATZ, BE BATTEIGER, RB JONES (Indianapolis, USA). *Sex Transm Dis* 1987;**14**:160–4.

Test-of-cure analysis by direct immunofluorescence for *Chlamydia trachomatis* after antimicrobial therapy
I NACHAMKIN, K SAWYER, D SKALINA, GW CROOKS, R CIOTTI, SJ SONDHEIMER (Philadelphia, USA). *J Clin Microbiol* 1987;**25**:1774–5.

In vitro activity of nonoxynol-9 on McCoy cells infected with *Chlamydia trachomatis*
ST KNIGHT, SH LEE, CH DAVIS, DR MOORMAN, RL HODINKA, PB WYRICK (Chapel Hill, USA). *Sex Transm Dis* 1987;**14**:165–73.

Non-specific genital infection and related disorders (mycoplasmal and ureaplasmal infections)

Humoral immune response to polypeptides of *Ureaplasma urealyticum* in women with postpartum fever
GY LEE, GE KENNY (Seattle, USA). *J Clin Microbiol* 1987;**25**:1841–4.

Non-specific genital infection and related disorders (general)

Detection of leukocyte esterase in urine: a new screening test for nongonococcal urethritis compared with two microscopic methods
M VEERAVAHU, RW SMYTH, JC CLAY (Birmingham, England). *Sex Transm Dis* 1987; **14**:180–4.

Pelvic inflammatory disease

Intrauterine devices and pelvic inflammatory disease: recent developments
DA GRIMES (Los Angeles, USA). *Contraception* 1987;**36**:97–109.

Pelvic inflammatory disease: bacteriology and sequelea
L WESTRÖM (Lund, Sweden). *Contraception* 1987;**36**:111–28.

Pelvic inflammatory disease associated with *Neisseria gonorrhoeae* and *Chlamydia trachomatis*: clinical correlates
BA CROMER, FP HEALD (Baltimore, USA). *Sex Transm Dis* 1987;**14**:125–9.

A microbiologic and clinical study of placental inflammation at term
Y DONG, PJSt CLAIR, I RAMZY, KS KAGAN-HALLET, RS GIBBS (San Antonio, USA). *Obstet Gynecol* 1987;**70**:175–82.

Single-agent therapy for women with acute polymicrobial pelvic infections
DL HEMSELL, MC HEARD, BJ NOBLES, PG HEMSELL (Dallas, USA). *Am J Obstet Gynecol* 1987;**157**:488–90.

Trichomoniasis

Detection of *Trichomonas vaginalis* antigen in women by enzyme immunoassay
A YULE, MCA GELLAN, JD ORIEL, JP ACKERS (London, England). *J Clin Pathol* 1987; **40**:566–8.

***Trichomonas vaginalis* NYH286 phenotypic variation may be coordinated for a repertoire of trichomonad surface immunogens**
JF ALDERETE (San Antonio, USA). *Infect Immun* 1987;**55**:1957–62.

Candidiasis

High-frequency switching in *Candida* strains isolated from vaginitis patients
DR SOLL, CJ LANGTIMM, J McDOWELL, J HICKS, R GALASK (Iowa, USA). *J Clin Microbiol* 1987;**25**:1611–22.

Evaluation of a new slide latex agglutination test for diagnosis of vaginal candidosis
V HOPWOOD, DW WARNOCK, JD MILNE, T CROWLEY, CT HORROCKS, PK TAYLOR (Bristol, England). *Eur J Clin Microbiol* 1987;**6**:392–4.

Genital herpes

Recurrences after oral and genital herpes simplex virus infection: influence of site of infection and viral type
WE LAFFERTY, RW COOMBS, J BENEDETTI, C CRITCHLOW, L COREY (Seattle, USA). *N Engl J Med* 1987;**316**:1444–9.

The purpose of this study was to evaluate the influences of anatomical site and viral type on recurrence patterns in oral and genital herpes simplex virus (HSV) infections. The prospective follow up of a cohort of young adults was undertaken. They all had concurrent oropharyngeal and genital primary HSV infections.

Thirty nine patients, whose history indicated that they were having a first episode of symptomatic genital herpes, were enrolled. At the initial clinic visit, patients had standard interviews about their histories and were examined, and specimens from the external genitalia, urethra, throat, and oral mucosa were obtained for culture. The women also had endocervical specimens taken for culture. Subsequent follow up was meticulous, with thrice weekly attendances for clinical examination and further viral sampling, until all lesions cleared. Viral serology was performed at enrolment and at the visit on day 21. HSV was isolated in diploid fibroblasts. Cell aliquots and hypernatant fluid from cultures where a cytopathic effect of HSV had been established were frozen at −70°C. The HSV isolates from first and subsequent clinic visits, from both oral and genital sites, were typed with monoclonal antibodies in a direct immunofluorescence assay. Restriction endonuclease analysis of viral DNA was performed to identify viral strains, in which at least four enzymes were used for each isolate, and at least 100 restriction enzyme sites evaluated for each isolate. Strain similarity was stringently defined as less than 1·7% variability in the number of restriction endonuclease sites between isolates. In the statistical analysis, comparisons between viral types regarding signs and symptoms of the primary episode were performed with the Wilcoxon rank sum or χ^2 tests. Recurrence rates were compared with the Wilcoxon test. The results showed impressive differences between recurrence patterns of HSV infection according to viral type and anatomical site. Orolabial recurrences developed in five of 12 patients with HSV-1 and one of 27 patients with HSV-2 ($p > 0{\cdot}001$). Conversely genital recurrences were seen in 24 of 27 patients with HSV-2 and three of 12 patients with HSV-1 ($p < 0{\cdot}01$). The mean frequency of subsequent

genital recurrences (due to HSV-1 and HSV-2) was 0·23 a month, whereas the mean frequency of oral or labial recurrences was only 0·04 a month ($p < 0·0001$). The mean monthly frequencies of recurrence were, in descending order, genital HSV-2 infections 0·33; oral or labial HSV-1 infections 0·12; genital HSV-1 infections 0·020; and oral HSV-2 infections 0·001 ($p < 0·01$ for each comparison). The authors concluded that the likelihood of reactivation of HSV infection differed between HSV-1 and HSV-2 infections, and between the trigeminal and sacral anatomical sites. Finally, they speculated that the fact that genital HSV infections recurred six times more often than oral or labial HSV infection might account (in the United States) for the relatively rapid increase in the prevalence of clinically recognised herpes in recent years.

G Sharp

Evaluation of an enzyme immunoassay for detection of herpes simplex virus antigen in genital lesions

J van ULSEN, AM DUMAS, JHT WAGENVOORT, A van ZUUREN, T van JOOST, E STOLZ (Rotterdam, The Netherlands). *Eur J Clin Microbiol* 1987;**6**:410–3.

Therapy of genital herpes with topically applied interferon

LJ ERON, C TOY, B SALSITZ, RR SCHEER, DL WOOD, PI NADLER Annandale, USA). *Antimicrob Agents Chemother* 1987;**31**:1137–9.

Genital warts

Human papillomavirus infections in women with and without abnormal cervical cytology

EM de VILLIERS, D WAGNER, A SCHNEIDER, *et al* (Heidelberg, Federal Republic of Germany). *Lancet* 1987;ii:703–6.

DNA typing of genital warts and diagnosis of sexual abuse of children

KA FLEMING, V VENNING, M EVANS (Oxford, England). *Lancet* 1987;ii:454.

Integration of human papillomavirus type 16 DNA sequences: a possible early event in the progression of genital tumors

S SCHNEIDER-MAUNOURY, O CROISSANT, G ORTH (Paris, France). *J Virol* 1987;**61**: 3295–8.

Natural killer cells in cervical intraepithelial neoplasia and human papillomavirus infection

SK TAY, D JENKINS, A SINGER (London, England). *Br J Obstet Gynaecol* 1987; **94**:901–6.

Patients with condyloma acuminatum exhibit decreased interleukin-2 and inteferon gamma production and depressed natural killer activity

R CAUDA, SK TYRING, CE GROSSI, *et al* (Birmingham, USA). *J Clin Immunol* 1987;**7**:304–10.

Trichloroacetic acid in the treatment of human papillomavirus infection of the cervix without associated dysplasia

VK MALVIYA, G DEPPE, R PLUSZCZYNSKI, G BOIKE (Detroit, USA). *Obstet Gynecol* 1987;**70**:72–4.

Malviya *et al* describe the use of 85% trichloroacetic acid for treating human papillomavirus (HPV) infection of the cervices of 48 patients (diagnosed by colposcopy and cytological change), who had no evidence of dysplasia and no cervical warts present on macroscopic examination. Coexistent vaginal and vulval warts were also treated simultaneously.

Patients were followed up with regular two week colposcopy to detect infection with HPV and were given up to four applications of trichoroacetic acid. Colposcopic and cytological evidence of HPV infection disappeared in 18 out of the 48 patients after one application of trichloroacetic acid for a follow up period of up to four months. Twenty eight patients required multiple applications of trichloroacetic acid. Seven patients had persistent disease despite up to three applications of trichloroacetic acid, and required laser ablation. Patients are being kept under continued follow up to detect recurrence of HPV changes on the cervix. During the course of the study patients were instructed to use condoms as far as possible throughout the treatment period so as to reduce the risk of infection from their male partners.

This report is interesting as it investigates a method of treatment that is cheaper and more readily applied than laser therapy. Prospective follow up of these patients will be particularly interesting, as it is in patients who have had laser treatment of the cervix. Recurrence of cervical HPV infection and associated changes, both cytologically and colposcopically, could arise from either the failure of treatment to eradicate human papillomavirus from basal and surface cells of the squamous epithelium of the cervix or from infection of the cervix from other areas of the genitourinary tract, such as the vagina, that are subclinically infected with HPV.

DJ Timmins

Topical self-treatment of penile warts with 0·5% podophyllotoxin in ethanol for four or five days

G von KROGH (Stockholm, Sweden). *Sex Transm Dis* 1987;**14**:135–40.

Acquired immune deficiency syndrome

AIDS and tuberculosis

KP GOLDMAN (Dartford, England). *Br Med J* 1987;**295**:511–2.

Ophthalmic findings in a group of ambulatory patients infected by human immunodeficiency virus (HIV): a prospective study

RC HUMPHRY, JN WEBER, RJ MARSH (London, England). *Br J Ophthalmol* 1987;**71**:565–9.

Adrenocortical function in acquired immunodeficiency syndrome

L MEMBRENO, I IRONY, W DERE, R KLEIN, EG BIGLIERI, E COBB (San Francisco, USA). *J Clin Endocrinol Metab* 1987;**65**:482–7.

Anterior ischaemic optic neuropathy in the acquired immune deficiency syndrome

MJ BRACK, PG CLELAND, RI OWEN, ED ALLEN (Sunderland, England). *Br Med J* 1987;**295**:696–7.

Epithelioid angiomatosis: a distinct vascular disorder in patients with the acquired immunodeficiency syndrome or AIDS-related complex

CJ COCKERELL, MA WHITLOW, GF WEBSTER, AF FRIEDMAN-KIEN (New York, USA). *Lancet* 1987;ii:654–6.

Perforating granuloma annulare in a patient with acquired immunodeficiency syndrome: immunohistologic evaluation of the cellular infiltrate

CJ HUERTER, J BASS, WF BERGFELD, RJ TUBBS (Cleveland, USA). *Arch Dermatol* 1987;**123**:1217–20.

Absence of Langerhans cells in oral hairy leukoplakia, an AIDS-associated lesion

TE DANIELS, MWSD GREENSPAN, JS GREENSPAN, *et al* (San Francisco, USA). *J Invest Dermatol* 1987;**89**:178–82.

Paraproteinemias in homosexual men with HIV infection: lack of association with abnormal clinical or immunologic findings

RM CRAPPER, DR DEAM, IR MACKAY (Melbourne, Australia). *Am J Clin Pathol* 1987;**88**:348–51.

Human immunodeficiency virus infection and routine childhood immunisation

CF von REYN, CJ CLEMENTS, JM MANN (Geneva, Switzerland). *Lancet* 1987;ii:669–72.

Perinatal infection with human immunodeficiency virus: specific antibody responses by the neonate

KH PYUN, HD OCHS, MTW DUFFORD, RJ WEDGEWOOD (Seattle, USA). *N Engl J Med* 1987;**317**:611–4.

Long latency precedes overt seroconversion in sexually transmitted human-immunodeficiency-virus infection

A RANKI, S-L VALLE, M KROHN, *et al* (Helsinki, Finland). *Lancet* 1987;ii:589–93.

Ranki *et al* describe the delayed appearance (seroconversion) of antibody to immunodeficiency virus (HIV) in patients from a cohort largely of Finnish homosexual men. (In the context of their work, "seroconversion" means becoming positive by a first generation enzyme linked immunosorbent assay (ELISA)). The patients were selected as follows: in a cohort of 235 men and two women, 23 initially had antibodies to HIV and nine seroconverted during 24 to 36 months. Stored sera from the nine seroconverters were studied retrospectively. Of the seronegative subjects in the cohort, 25 whose sexual partners were seropositive, nine with lymphadenopathy, and 14 who were symptomless were selected for prospective study. Serum samples from the subjects were tested for HIV p24 antigen and for antibodies to HIV by first generation ELISA, by competitive ELISA for antibodies to core and envelope recombinant HIV proteins, and by western blotting. Lymphocytes were tested by in situ hybridisation tests under high stringency for HIV sequences using an HTLV-III_B specific RNA probe. They were also tested in vitro for response to PPD (tuberculin purified protein derivative).

Of the nine subjects who seroconverted, eight showed an isolated partial response to antibody HIV by western blot to p17, p24, or p55 in successive samples collected 7–14 months previously. In some these antibodies were IgM and in some IgG. The titres against these core proteins were low, 1/50 to 1/200 compared with 1/800 to 1/12800 after seroconversion. Further tests with recombinant core, envelope, 3′ *orf*, *sor*, and *tat* proteins of HIV as antigens showed antibody in all nine subjects before seroconversion. Of 110 control samples similarly tested, only one had antibody to HIV p24 (by western blot only). Two of the nine seroconverters were also HIV antigen positive at some point before seroconversion.

Of the 25 seronegative sexual partners of seropositive men and the 23 others, seven had signs of latent HIV infection, mainly fluctuating HIV antigen during follow up for up to 34 months. Five seronegative subjects had weak antibodies to core by western blot. In five of the nine seroconverters absent or reduced in vitro proliferative response to PPD had been seen 6–14 (mean 10) months previously. This also occurred in three of the seven seronegative subjects who had some HIV markers. Two of three latently infected seronegative subjects had evidence of HIV RNA in mononuclear cells (<0·01% of adherent cells). Four of the nine seroconverters had a mild primary illness and two had developed lymphadenopathy in the latent phase. Three other latently infected subjects had mild lymphadenopathy. The authors suggest that the latent period that they describe, with prolonged weak and partial response to HIV, may be due to HIV being tropic for antigen presenting cells which, though infected, fail to trigger a full antibody response.

This report, especially if its findings are confirmed, is of considerable importance for understanding early events in HIV infection and for managing patients at risk of infection. Many patients seen at clinics might have latent infections similar to those described but be falsely reassured by negative results to conventional tests for antibody to HIV; such patients might be infectious (HIV antigen was found in some of the study patients, though no report is made of HIV isolation studies). There are some reasons for doubting whether the findings presented are representative of the normal pattern of HIV infection. Firstly, seroconversion after single exposures to HIV, such as blood transfusion and organ transplantation, has been within 1–2 months. Observations on patients with haemophilia have shown delayed seroconversion, however, and the length of time before seroconversion may depend critically on dose. Secondly, the specificity of the assays used are not known. False positive readings have been recognised in some western blots, and antibodies to single recombinant proteins have not been widely tested for elsewhere. Thirdly, other workers have used HIV antigen tests and other sensitive tests (specific IgM, second generation ELISA) for early HIV infection in large numbers of people at risk, including regular sexual partners of seropositive men, but have not had the positive results reported here. Fourthly, no successful HIV isolations are reported in the latent phase described.

These things said, it must be admitted that other investigators have neglected to record defined exposure events in their studies of patients sexually at risk, have tended to ignore the evidence from treatment of haemophiliacs, and have neglected other general evidence from studies of retroviruses, such as HTLV-I and bovine leukaemia virus, that seroconversion may be delayed from a "normal" interval of a few weeks to many months. The possibility that this may be common after sexual exposure to small doses of HIV cannot be ignored. New, highly sensitive, and commercially available tests for HIV antigen and antibody to HIV should now be applied to groups of patients chronically exposed to seropositive partners, especially those with lymphadenopathy. Patients with histories of exposure well defined temporally are also an important group for study. Vigorous efforts should be made to isolate HIV from both. It is just possible that if a long latent stage of HIV infection precedes overt seroconversion, prompt intervention with a drug such as azidothymidine (AZT) might entirely reverse the infectious process. Such a prospect, as well as the management problems raised by HIV latency, demands that this matter receives intensive study.

PP Mortimer

Infection with the human immunodeficiency virus: clinical manifestations and their relationship to immune deficiency: a report from the multicenter AIDS cohort study

RA KASLOW, JP PHAIR, HB FRIEDMAN (Bethesda, USA). *Ann Intern Med* 1987;**107**:474–80.

Risk of AIDS related complex and AIDS in homosexual men with persistent HIV antigenaemia

F de WOLF, J GOUDSMIT, DA PAUL, *et al* (Amsterdam, The Netherlands). *Br Med J* 1987;**295**:569–72.

Correlation of enzyme-linked immunosorbent assays for serum human immunodeficiency virus antigen and antibodies to recombinant viral proteins with subsequent clinical outcomes in a cohort of asymptomatic homosexual men

KH MAYER, LA FALK, DA PAUL, *et al* (Pawtucket, USA). *Am J Med* 1987;**83**:208–12.

Temporal relation of antigenaemia and loss of antibodies to core antigens to development of clinical disease in HIV infection

C PEDERSEN, CM NIELSEN, BF VESTERGAARD, J GERSTOFT, K KROGSGAARD, JO NIELSEN (Hvidovre, Denmark). *Br Med J* 1987;**295**:567–9.

Unique p24 epitope marker to identify multiple human immunodeficiency virus variants in blood from the same individuals
J BOLTON, NC PEDERSEN, J HIGGINS, M JENNINGS, J CARLSON (Davis, USA). *J Clin Microbiol* 1987;**25**:1411–5.

Detection of human immunodeficiency virus core protein in plasma by enzyme immunoassay: association of antigenaemia with symptomatic disease and T-helper cell depletion
AE WITTEK, MA PHELAN, MA WELLS, *et al* (Bethesda, USA). *Ann Intern Med* 1987;**107**:286–92.

Current concepts in the virology of infection with human immunodeficiency virus (HIV): a view from the III international conference on AIDS
A HOXIE (Philadelphia, USA). *Ann Intern Med* 1987;**107**:406–8.

Pathogenesis of infection with human immunodeficiency virus
DD HO, RJ POMERANTZ, JC KAPLAN (Los Angeles, USA). *N Engl J Med* 1987;**317**:278–86.

Persistent infection of chimpanzees with human immunodeficiency virus: serological responses and properties of re-isolated viruses
PL NARA, WG ROBEY, LO ARTHUR, *et al* (Frederick, USA). *J Virol* 1987;**61**:3173–80.

Functional regions of the envelope glycoprotein of human immunodeficiency virus type 1
M KOWALSKI, J POTZ, L BASIRIPOUR, *et al* (Boston, USA). *Science* 1987;**237**:1351–5.

Immunology of human immunodeficiency virus infection and the acquired immunodeficiency syndrome: an update
M SELIGMANN, AJ PINCHING, FS ROSEN, *et al* (Geneva, Switzerland). *Ann Intern Med* 1987;**107**:234–42.

Immunology of infection with the human immunodeficiency virus (HIV): a view from the III international conference on AIDS
S KOENING, ZF ROSENBERG (Bethesda, USA). *Ann Intern Med* 1987;**107**:409–12.

Diagnosis of human immunodeficiency virus infection in seronegative homosexuals presenting with an acute viral syndrome
HA KESSLER, B BLAAUW, J SPEAR, DA PAUL, LA FALK, A LANDAY (Chicago, USA). *JAMA* 1987;**258**:1196–9.

Isolation of acquired immunodeficiency syndrome virus from the placenta
WC HILL, V BOLTON, JR CARLSON (Sacramento, USA). *Am J Obstet Gynecol* 1987;**157**:10–1.

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Microbiologic and serologic studies of *Gardnerella vaginalis* in intra-amniotic infection
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In this study the authors used quantitative cultures and antibody response to assess the role of *Gardnerella vaginalis* in intra-amniotic infections. Amniotic fluid was collected from patients with clinical intraamniotic infection and asymptomatic controls by a transcervical pressure catheter. The authors excluded the possibility of contamination by discarding the first 7 ml of the sample. The specimen was innoculated on media for aerobes, anaerobes, mycoplasmas, and V agar selective (biphasic blood agar) organisms. Serum was collected within two days of delivery (acute phase) and six weeks later (convalescent phase). Antibody response was measured by microenzyme linked immunosorbent assay. Of 300 samples, 86 were selected from patients with intra-amniotic infection and 86 from symptomless patients. The mean gestational age was 39·4 weeks. The mean interval from rupture of membrane to specimen collection was 16·6 hours. The isolation of *Gardnerella vaginalis* from the amniotic fluid of infected or symptomless women did not differ significantly.

G vaginalis was found commonly in association with mycoplasmas. Paired serum samples from unmatched but similar groups of patients, 25 with intraamniotic infections (eight *G vaginalis* positive, and 17 negative) and 18 symptomless (seven *G vaginalis* positive, and 11 negative), were tested. Mean antibody concentrations were found to be similar at the acute phase. No significant differences in antibody concentrations were found between patients aged under 20 and those aged 20–44.

The authors concluded that their data do not support a pathogenic role of *G vaginalis* in intraamniotic infection. The study does not, however, exclude a facilitating role of *G vaginalis* in polymicrobial infection.

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Reduction of group B streptococcal maternal and neonatal infections in preterm pregnancies with premature rupture of membranes through a rapid identification test
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Longitudinal study of group B streptococcal carriage during late pregnancy
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Neonatal sepsis with or without meningitis has long been known to be a major cause of perinatal mortality. That due to *Escherichia coli* has been attributed to vertical transmission from the maternal gastrointestinal tract. The maternal source of group B streptococcal septicaemia has been postulated to be the genitourinary tract or, more recently, the gastrointestinal tract.

From September 1983 to March 1984 Persson *et al* studied 152 pregnant women from the 37th week of pregnancy to delivery. Their aim was to assess whether the rectum, urethra, or urine would provide the best index of chronic carrier status of group B streptococci, thus indicating which mothers and children would benefit from chemoprophylaxis at delivery. Their figures appear to support the theory that the rectum may be a chronic reservoir for group B streptococci, as 68% of those defined as being chronic carriers (yielding three consecutive positive cultures) had positive rectal cultures. As Persson *et al* point out, however, the numbers of women studied were too small to allow conclusive statistical calculations. They also point out that chemoprophylaxis of all carriers of group B streptotocci at delivery may produce bacterial resistance more often than prevent infection.

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